CASE SERIES JSLS

Late Hemorrhage Following Laparoscopic Cholecystectomy

Rajeev Sinha, MS, MD, Arun Gupta, MD

ABSTRACT

Background: Excruciating generalized abdominal pain with features suggestive of shock, at the end of the first or early second week after laparoscopic cholecystectomy (LC), is a frightening and formidable diagnostic predicament. This is because the early known complications like biliary leak or vascular injuries are unlikely diagnoses. Hemoperitoneum, is not usually considered, but instead more common occurrences like acute pancreatitis, choledocholithiasis, and sepsis are suspected. A delay in diagnosis and subsequent management of hemoperitoneum could have disastrous consequences.

Case Studies: Two patients presented with hemoperitoneum, in the second week after laparoscopic cholecystectomy. The first was because of a leak from a pseudoaneurysm of the right hepatic artery and the other was a bleed from a subcapsular liver hemangioma as a part of Osler Weber Rendu syndrome. Initially, a clinical assessment in both the patients was diagnostically inconclusive. Ultimately the diagnosis could be made, based on computed tomography angiography and visceral angiography. In the second patient, a positive family history and genetic testing were helpful. The first patient was successfully managed by intravascular embolization, while the second patient was successfully managed conservatively with intraperitoneal drains and conservative management of comorbidities.

Conclusions: The presentation is to generate awareness that hemorrhage could be a presentation, in the early second week, after LC. A common cause to be considered is a pseudo aneurysmal bleed. Secondary hemorrhage and other rare coincidental unassociated conditions could also be responsible for the hemorrhage. A high index of suspicion, and early and timely management are keys to a successful outcome.

Key Words: Angioembolization, Hemoperitoneum, Hepatic artery pseudoaneurysm, Laparoscopic cholecystectomy complication, Osler Weber Rendu syndrome.

INTRODUCTION

A sudden acute abdominal pain, with features of shock, in the second postoperative week after laparoscopic cholecystectomy (LC), can be a shocking and formidable enigma for any surgeon. Especially so if the postoperative course had been uneventful up to this point. While analyzing the probable reasons, the time of presentation becomes important because the second week post LC, is the time when most of the common, acute immediate postoperative complications, like early biliary leak or reactionary hemorrhage are no longer under consideration as likely causes. Instead, other causes like post-LC acute pancreatitis, cholangitis with or without choledocholithiasis, late biliary leaks, or more likely septic shock, need to be entertained. But hemoperitoneum is usually not considered as a differential diagnosis in the second postoperative week, especially if the initial postoperative course had been uneventful. Although sepsis induced secondary hemorrhage may occur but it will be associated with preceding, obvious signs and symptoms of infection. The sudden hemoperitoneum, if it occurs, can be either because of very uncommonly encountered post-LC complication of a bleeding aneurysm or pseudoaneurysm (PsA) of the hepatic artery (PsA-HA) or right hepatic artery (PsA-RHA),[1,2] a large abdominal wall hematoma,[3] or even

Department of General and Laparoscopic Surgery, MLB Medical College, Jhansi, India. (Dr. Sinha)

Department of Interventional Radiology, Sir Ganga Ram Hospital, New Delhi, India. (Dr. Gupta)

Acknowledgements: none.

Disclosure: none.

Conflict of interests: none.

Funding sources: none.

Informed consent: Dr. Rajeev Sinha declares that written informed consent was obtained from the patient/s for publication of this study/report and any accompanying images.

Address correspondence to: Dr. Rajeev Sinha, MS, MD, Sinha Villa, New 187 (Old57), Civil Lines, Jhansi, UP, India, 284128, Telephone: +919415030006, Email: sinha_rga@yahoo.co.in.

DOI:10.4293/JSLS.2023.00006

because of an absolutely unrelated coincidental disease, as was seen in our second patient.

We report two cases of postoperative LC patients, with intraabdominal bleeds and shock, early in the second postoperative week. The first was because of a PsA bleed on postoperative day (POD) 8 and the second was a POD 10 bleed because of a coexisting hereditary-hemorrhagic telangiectasia (HHT) syndrome, unknown both to the patient and the operating team. The unexpected presentation could be diagnosed with great difficulty especially the latter one, but both were successfully managed. The need to draw attention to these two cases as possible causes of late post-LC hemoperitoneum, is to raise an index of suspicion. Early and timely diagnosis and management, would help in reducing morbidity and even mortality, significantly.

CASE PRESENTATIONS

Case 1

A 35-year-old female was diagnosed with gall stones after an abdominal ultrasound and underwent uneventful LC for cholelithiasis. The approach used the standard 3 ports, a 10 mm infraumbilical, a 5 mm epigastric, and a third 5 mm subcoastal midclavicular line port. The dissection was done with a harmonic scalpel. The cystic artery was short and was ligated, while the cystic duct was clipped. The patient underwent uneventful LC for cholelithiasis and was discharged on POD 2. Her pre-operative profile and parameters were all within normal range. At the time of discharge she had mild abdominal gaseous discomfort with paucity of flatus.

She developed recurrent mild colicky pain on POD 6. The pain was localized in the upper abdomen. There was gaseous distension and paucity of flatus and feces. Clinically the patient did not have fever or jaundice and her complete blood count (CBC), white blood cell count (WBC), and serum bilirubin and liver enzymes, including aspartate transferase (AST) and alanine transaminase (ALT) were within normal range. She responded to conservative drug therapy. Two days later on POD 8, she developed excruciating pain in the upper abdomen, immediately followed by hypotension and tachycardia.

At the time of admission she had a blood pressure (BP) of 80 mm Hg systolic and a pulse rate (PR) of 122/min. Extremities were cold, radial pulse was feeble, and abdomen had generalized tenderness. Acute pancreatitis or sepsis were considered as possibilities and investigated accordingly. Her investigations showed, a very low hemoglobin (Hb) at 6.8 gm/dl, CBC 20,700 cells/per mm^3, serum bilirubin was 0.72 mg %, AST 18.2 U/L, ALT 17.9 U/L, alkaline phosphatase (ALP) 100 IU/L, C-reactive protein 22 mg/L, and procalcitonin (PCT) 0.75ug/L. Serum lipase and amylase were 96 U/L and 198 U/L, respectively. The coagulation profile was within normal range. An ultrasound of the abdomen and noncontrasted computed tomography (NCCT) of the abdomen showed a large collection in the upper abdomen with moderate ascites. The patient was resuscitated with fluids and 4 bottles of whole blood. An ultrasound guided diagnostic abdominal paracentesis was negative. Eighteen hours post-resuscitation parameters showed, Hb 10.4 mg %, TLC 24,400 cells per mL, serum bilirubin was 1.04 mg %, s. creatinine was 1.34 mg %, and serum sodium and potassium were 134.5 mEq/L and 3.59 mEq/L, respectively.

The next day, POD 9, the patient had a repeat laparoscopy. On the second laparoscopy, the supracolic compartment was organized with clots and all organs were adherent. The hematoma and clots were maximum at the splenic flexure and left paracolic gutter. There was no trace of any bile, and no active source of bleed could be found. Further exploration was not possible and was not attempted. The patient remained stable with PR 120/min, BP at 110/60 mm Hg, respiration rate 24/minute, and she was conscious and maintaining her oxygen concentration.

She was transferred to another center on POD 9. A digital subtraction angiogram of the common hepatic artery showed pseudoaneurysm arising from the right hepatic artery involving right anterior and posterior hepatic artery origins. The clips can be seen in close vicinity of the PsA (**Figure 1**). Endovascular embolization with glue and lipiodol was successfully achieved and postembolization angiogram showed nonopacification of pseudoaneurysm (filled with glue cast) with preserved hepatic arteries (**Figure 2**). The patient rapidly responded to conservative treatment and was discharged 4 days later and is now normal.

Case 2

A 61-year-old female with controlled Type 2 diabetes, and controlled hypertension for the last 20 years, underwent an uneventful laparoscopic cholecystectomy and was discharged the second day. She had a history of multiple operations: two lower segment cesarean sections, tubal ligation 25 years ago, and

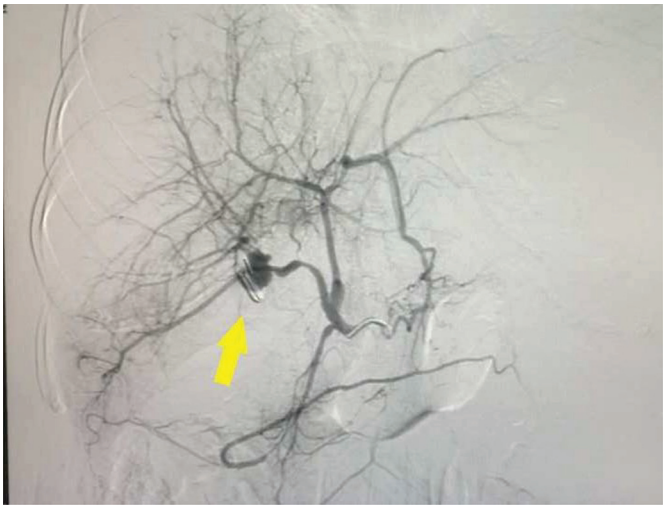

Figure 1. Digital substraction angiogram of common hepatic artery shows pseudoaneurysm arising from right hepatic artery involving right anterior and posterior hepatic artery origins. Clips can be seen in close contact.

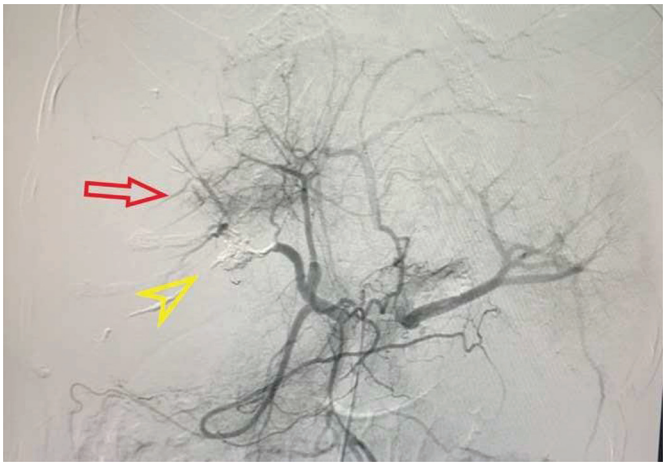

Figure 2. Post glue and lipiodol embolization angiogram shows nonopacification of pseudoaneurysm, filled with glue cast (yellow arrow) and with preserved hepatic arteries (red arrow).

hysterectomy 15 years ago. She also had history of spontaneous attacks of epistaxis, almost every month with spontaneous resolution or after cold compresses, which was attributed to her hypertension and was previously investigated. Postoperatively, her pre-operative presenting complaints of abdominal discomfort and bloating persisted and was attributed to her prolonged diabetes related gastropathy. At the time of discharge her vitals were normal.

She experienced sudden severe epigastric pain and syncopal attack on POD 10. At the time of presentation, she was afebrile, had tachycardia >150/min, respiratory rate was 27/min, BP was 80/60 mm Hg, blood oxygen was 94%, and extremities were cold and clammy. Abdomen had generalized tenderness but rigidity was not noticeable. She was provisionally kept as acute pancreatitis and managed as such with fluid support. The BP responded to 120/60 mm Hg and tachycardia settled to 120/min. Her hemoglobin was 6.7 gm %, CBC was 11700/cu mm and 68.7% neutrophils. The platelet count was 2.56 lac/cu mm. Serum amylase and serum lipase were both within normal range, 70.0 U/L and 95.0 U/L, respectively. Hepatobiliary parameters, with serum bilirubin 1.07 and AST (26.0 IU/L), ALT (15.4 IU/L), and ALP (69.0 IU/L). Serum troponin to rule out a cardiac event was also normal at 0.018 ng/ml. Serum sodium (137.3 mEq/L) was within range but serum potassium was slightly raised (5.68 mEq/L). Procalcitonin was also within range at 0.15ug/L. Further blood tests of the coagulation profile showed mildly raised INR (1.41); APTT, BT, CT were all within normal range as also the TEG values. The reports ruled out acute pancreatitis, sepsis, and possible biliary peritonitis. An emergency NCCT and ultrasound of abdomen showed collection in the whole peritoneal cavity, more on the right side and was diagnosed as hemoperitoneum. An ultrasound-guided, diagnostic peritoneal paracentesis ruled out biliary peritonitis and showed hemorrhagic fluid.

A CT angiogram showed three splenic artery aneurysms at the hilum and a 4.0 × 4.8 cm sub-capsular hepatic hemangioma segment V and VI, but there was no active contrast extravasation. In the absence of extravasation of dye, it was evident that the bleed had stopped spontaneously. The likely source was attributed to a leak from one of the aneurysms or the subcapsular liver hemangioma. She was transfused 2 units of whole blood and her hemoglobin improved to 7.9, TLC 14,200, amylase 150.

She was transferred to a referral medical center, where she was reinvestigated and was kept under observation. Two days later, she developed ecchymotic patches over her trunk and tenderness and distension of abdomen was more marked in epigastrium and right hypochondrium. Fresh investigations for consumptive coagulopathy (D-dimer, fibrin degradation products) and procalcitonin were again found to be normal. Meanwhile she also developed bilateral pleural effusion, approximately 100 – 120 ml. Her serum bilirubin and liver enzymes remained in normal range.

At this time, we learned of the patient's family history of Osler Weber Rendu syndrome, specifically her brother, and she was referred for genetic consultation. She was having tachypnea and upper abdominal pain more marked with respiration. Using ultrasound guidance, percutaneous catheter drains were inserted in both hypochondrium, which drained 300 ml and 200 ml of fluid on the first day. Three hundred ml of right pleural effusion was also aspirated. She was given 2 units of red blood cell transfusions and 8 units of plasma and supportive therapy as required. She was discharged after 20 days and has now recovered.

DISCUSSION

Incidence of isolated vascular injuries, associated with LC may occur in almost 0.8% of patients[2,4], and in association with biliary ductal injury in as many as 25% of patients.[5,6] Among the vascular injuries, the per operative or reactionary bleeds are relatively easy to diagnose, but later bleeds, as in early second week, can occur from pre-existing aneurysms or PsA of the biliary vascular tree, and require a high index of suspicion for diagnosis. Incidentally, PsA of the hepatic artery (HA) account for almost 50% of all HA aneurysms.[4]

The late post-LC bleed has most commonly been attributed to a PsA-RHA in 60% – 70% of cases or less commonly, the common hepatic artery in 30% of cases, cystic A in 10% of cases, or even gastroduodenal artery.[1,8,9]

PsA-RHA can result after any infective pathology, like acute or chronic cholecystitis,[10–12] or pancreatitis,[9] infected bilioma,[13,14] liver abscesses, or after interventions in the hepatobiliary area, like post-liver transplantation, PCT aspirations, biopsies or cholangiography, or liver trauma.[2,9] Association has also been shown between PsAs and anatomical variations of the hepatobiliary vasculature and ductular systems.[16,17] The most common cause for PsA-RHA is after iatrogenic trauma during LC.[9] Such iatrogenic trauma is more common during difficult LC and has been reported in as many as 3 in 100,000 LCs for acute cholecystitis and in 1 in 100,000 of emergency LCs.[2]

It can manifest as a delayed complication after an operatively repaired HA injury.[18] In the absence of an obvious HA injury, the possible pathophysiological events leading to formation of PsA after LC, are still unknown but hypothetically it can be because of direct, incomplete injury, to the arterial wall due to dissipating energy from a laser or monopolar electrical energy source.[8,19] Alternately, it could be due to indirect conduction through a clip onto the vessel wall,[2,8,20] although this latter hypothesis, seems less likely considering that clips are the last to be applied, after energy aided dissection in the Calot's triangle is complete.

Lateral thermal spread from the point of application of the energy source is always an important consideration. In this regard the minimal lateral dissipation of energy is with the use of bipolar electrosurgical current followed by an ultrasonic shear. Bipolar cautery or ultrasonic dissectors may be less likely to damage the vessel wall by virtue of less carbonization,[36] especially when used for thick gall bladders or intrahepatic gall bladders.[2]

The use of monopolar current is associated with much more lateral spread, while the use of laser leads to maximum lateral damage. The charring weakens the vessel wall, which then dilates. The charred patch sheds, after a variable time, and to an extent promoted by the bile acids in the bile,[2,13,20] and leads to a leak, resulting in a localized collection and cavity identifiable as a PsA. The PsA in contrast to a localized hematoma is still in contact with the arterial lumen, while the latter is not.[3] Alternately, in contrast to the possible energy transfer through the clips, PsA-RHA or PsA-HA can also be a result of direct mechanical injury caused by clips on the cystic duct lying in close contact with and intruding on the segmental branches of HA[21,22], a hypothesis corroborated by the identification of clips adjacent to the PsA. In our first patient the clips can be seen lying in close vicinity of the PsA. (**Figure 1**).

PsA bleeds, have been mentioned across variable time intervals after LC and ranging from within 2 weeks,[15,19] most commonly within the first month,[23] to as late as 13 months after LC,[24] or even at 5 years post-LC.[9] Risk of rupture, has been variously mentioned as 21% to 80%,[2,15] and may be related to size, with almost tenfold increase with size >5 cm.[9] The morbidity and mortality, post-bleed, is as much as 35% – 50%.[1,2]

The rupture, when it occurs can be, into the peritoneal cavity, or most commonly into the extrahepatic biliary ductular channel especially where there is an associated ductular injury or even into the cystic duct stump.[25] Less commonly, rupture of the PsA can occur into the portal or hepatic venous system.[4] It can also rupture into parts of the gastrointestinal (GI) tract, with the duodenum or ileum presenting as GI bleed.[26–28]

Although the presentation of PsA could be an incidental asymptomatic finding, the most common presentation is with features of haemobilia, when the PsA ruptures into

the biliary channel, presenting as upper GI bleed or melena in as many as 85.1% patients.[9,15,17] Otherwise, the source of haemobilia is most commonly, HA in 88.1%, cystic A in 7.9%, and a combination of both in 4%.[29] The other manifestations are upper abdominal pain in 70% and jaundice in 60%.[2] Less than 40% present with Quincke's triad (jaundice, biliary colic, and GI bleed) described for the first time in 1857.[17,23] But, by far the most dangerous manifestation of rupture is hemoperitoneum, especially in late first or early second week. Small leaks get localized but more severe bleeding presents as hypovolemia and hemoperitoneum. When an initial small leak is soon followed by a much more severe bleed it has been termed as 'double rupture phenomena'.[9]

A simple ultrasound of the abdomen cannot be a diagnostic tool, except that it can indicate collections in the peritoneal cavity, and often differentiate a PsA from a hematoma.[30] Similarly a repeat laparoscopy confirms hemoperitoneum, an important differentiation from biliary peritonitis. An endoscopic retrograde cholangiopancreatography, or upper GI or lower GI endoscopy, can all contribute toward suggesting haemobilia, with blood in the proximal or distal bowel.[4] But the more specific and confirmatory investigations include, a color Doppler adjunct to a CT, occasionally showing swirling blood within PsA-HA, described as 'yin-yang' sign.[30] However multidetector CT angiography[31,32] and catheter angiography[33] are the best tools for diagnosis. The latter also has the advantage of being therapeutic.

The first line of treatment should be intra-arterial embolization of the PsA, if it is in communication with a patent vessel, and the success rate is up to 80%.[9] The embolization can be carried out using gelatin sponge, steel coil, cyano acrylate,[9] or thrombin.[34] Our patient had a successful PsA embolization using glue and lipiodol, and postembolization angiogram shows nonopacification of pseudoaneurysm (filled with glue cast) with preserved hepatic arteries (**Figure 2**). The procedure is repeatable, which is a distinct advantage. Other advantages include, the procedure is possible under local anesthesia, minimal liver parenchymal damage, reduced morbidity, and very early recovery. Embolization has its own complications, including rupture of the PSA during coil embolization, postembolization liver abscess,[35] hepatic ischemia, and bowel infarction by extension of thrombosis.[34] CBD stricture,[8,36] or a more common failure of embolization. Failure of embolization could be multifactorial[36,37] and can be supplemented with nonselective embolization of the HA or surgical exploration of the PsA-HA. Use of stents has also been reported in PsA with stenosis.[38] Surgical options include, exploration with excision of PsA-RHA with, RHA ligation,[39] or if the hepatic artery is involved, then repair with patch or venous or synthetic graft interposition. A block in RHA flow, as in some patients with embolization of the PsA where the thrombus extends beyond into the lumen, or after RHA ligation, does not usually result in liver parenchymal infarction. This is because of two reasons, one, the parenchyma continues to receive blood through the portal vein,[40] and two, arterial collateralization from the preserved branch of the HA.[40] Although, a much more formidable partial hepatectomies following ischemia have also been reported.[13,18,36] But post-treatment parenchymal vascular compromise, which may need partial hepatectomy, can also be avoided by proper evaluation of the RHA variations[39] and assessment of adequate arterial and collateral blood flow prior to embolization especially in an already compromised liver parenchyma, such as cirrhosis.[8,38]

The hemoperitoneum, in our second patient, was because of an unrelated cause, but occurred in the same period, and was much more difficult to diagnose. Even if abnormal angiographic findings are present, they may not necessarily be the cause of bleed. In such instances, only after exclusion of the other obvious causes of bleed, can an alternate diagnosis be looked for. This requires revisiting the patient's medical history, a detailed history for any drug intake, a history of any relevant coincidental disease, and a family history of any bleeding or clotting disorder. Also needed is a coagulation profile and knowledge of syndromic bleeding states that could fit the pattern of available clinical data of the patient. In the second patient, where after failure to find an obvious cause for bleeding, a positive family history of HHT was elicited. The angiographic findings were then explainable and the final confirmation was with genetic testing. It is beyond the scope of this article to discuss the vast number of likely unrelated causes but a high index of suspicion for such causes helps.

CONCLUSION

The two patients reported here highlight the fact that post-LC abdominal discomfort may not always be a minor event. Also when the post-LC patient presents at the end of the first week or early second week, with features of sudden excruciating abdominal pain, severe pallor, and shock, a diagnostic dilemma always exists. In such circumstances, a knowledge of, and a high index of suspicion, about less commonly occurring post-LC complications and in some instances causes absolutely unrelated to LC, may be of immense benefit for both the patient and the treating

team. For this a stepwise analysis is required and apart from the basic contrast enhanced CT, magnetic resonance imaging angiography, and invasive catheter angiography, are essential diagnostic tools. PsA are best managed endovascularly with a good success rate, minimal morbidity and mortality, and early recovery. Unrelated causes require appropriate treatment and may respond to conservative treatment as in this patient with Osler Weber Rendu syndrome, Also if the LC related causes are ruled out as the cause for hemoperitoneum, rare events should be kept in mind. Without a high index of suspicion, late post-LC hemorrhages may be diagnosed and treatment delayed, thus adding to morbidity and even mortality.

References:

1. Psathakis D, Müller G, Noah M, Diebold J, Bruch HP. Present management of hepatic artery aneurysms. Symptomatic left hepatic artery aneurysm; right hepatic artery aneurysm with erosion into the gallbladder and simultaneous colocholecystic fistula–a report of two unusual cases and the current state of etiology, diagnosis, histology and treatment. *Vasa.* 1992;21(2):210–215.

2. Sansonna F, Boati S, Sguinzi R, Migliorisi C, Pugliese F, Pugliese R. Severe hemobilia from hepatic artery pseudoaneurysm. *Case Rep Gastrointest Med.* 2011;925142.

3. Ahmed M, Fontecha C, Atchison M, et al. Post-cholecystectomy hepatic artery pseudoaneurysm rupture. *Cureus.* 2021;13(9):e18223.

4. Finley DS, Hinojosa MW, Paya M, Imagawa DK. Hepatic artery pseudoaneurysm: a report of seven cases and a review of the literature. *Surg Today.* 2005;35(7):543–547.

5. Pulitano C, Parks RW, Ireland H, Wigmore SJ, Garden OJ. Impact of concomitant arterial injury on the outcome of laparoscopic bile duct injury. *Am J Surg.* 2011;201(2):238–244.

6. Yelle JD, Fairfull-Smith R, Rasuli P, Lorimer JW. Haemobilia complicating elective laparoscopic cholecystectomy: a case report. *Can J Surg.* 1996;39(3):240–242.

7. Abbas MA, Fowl RJ, Stone WM, et al. Hepatic artery aneurysm: factors that predict complications. *J Vasc Surg.* 2003;38-(1):41–45.

8. Nakase Y, Takagi T, Fukumoto K, et al. Hemobilia and cystic artery stump pseudoaneurysm associated with liver abscess after laparoscopic cholecystectomy: report of a case. *Surg Today.* 2008;38(6):567–571.

9. Bulut T, Yamaner S, Bugra D, Akyuz A, Acarli K, Poy- Anli A. False aneurysm of the hepatic artery after laparoscopic cholecystectomy. *Acta Chir Belg.* 2002;102(6):459–463.

10. Akatsu T, Tanabe M, Shimizu T, et al. Pseudoaneurysm of the cystic artery secondary to cholecystitis as a cause of hemobilia: report of a case. *Surg Today.* 2007;37(5):412–417.

11. Saluja SS, Ray S, Gulati MS, Pal S, Sahni P, Chattopadhyay TK. Acute cholecystitis with massive upper gastrointestinal bleed: a case report and review of the literature. *BMC Gastroenterol.* 2007;7:12.

12. Lee JW, Kim MY, Kim YJ, Suh CH. CT of acute lower GI bleeding in chronic cholecystitis: concomitant pseudoaneurysm of cystic artery and cholecystocolonic fistula. *Clin Radiol.* 2006;61(7):634–636.

13. Madanur MA, Battula N, Setfli H, Desflpande R, Heaton N, Rela M. Pseudoaneurysm following laparoscopic cholecystectomy. *Hepatobiliary Pancreat Dis Int.* 2007;6:294–298.

14. Frilling A, Li J, Weber F, Fruhans NR, et al. Major bile duct injuries after laparoscopic cholecystectomy: a tertiary center experience. *J Gastrointest Surg.* 2004;8(6):679–685.

15. Khalil A, Fawzy FS, Zaid MH. Management of iatrogenic hepatic artery pseudoaneurysm *Egyptian Journal of Surgery.* 2021;40:954–958.

16. Masannat YA, Al-Naser S, Al-Tal Y, Al-Koteesfl J, Sflaraf UI. A rare complication of a common operation: Hepatic artery pseudo aneurysm following cholecystectomy report of a case. *Ir J Med Sci.* 2008;177(4):397–398.

17. Malik KA, Muneeb MD, Jawaid M, Kflan ML. Post laparoscopic cholecystectomy hepatic artery pseudo-aneurysm. *Pak J Surg.* 2010;26:89–91.

18. Singh P, Scibelli N, Gosal K, et al. Hepatic artery pseudoaneurysm presenting as Ggastrointestinal hemorrhage. *Cureus.* March 30, 2021;13(3):e14190.

19. Genyk YS, Keller FS, Halpern NB. Hepatic artery pseudoaneurysm and haemobilia following laser laparoscopic cholecystectomy: a case report. *Surg Endosc.* 1994;8(3):201–204.

20. Bin Traiki TA, Madkflali AA, Hassanain MM. Haemobilia post laparoscopic cholecystectomy. *J Surg Case Rep.* 2015(2):rju159.

21. Christensen TL, Matsuoka L, Heestand G, et al. Iatrogenic pseudoaneurysms of the extrahepatic arterial vasculature: management and outcome. *HPB (Oxford).* 2006;8(6):458–464.

22. Srinivasaiah N, Bhojak M, Jackson M, Woodcock S. Vascular emergencies in cholelithiasis and cholecystectomy: our experience with two cases and literature review. *Hepatobil Pancreat Diseases Int.* 2008;7(2):217–220.

23. Hewes JC, Baroni ML, Krissat J, Bhattacharya S. An unusual presentation of hepatic artery aneurysm as a complication of laparoscopic cholecystectomy. *Eur J Surg.* 2002;168:566–568.

24. Milburn JA, Hussey JK, Bachoo P, Gunn IG. Right hepatic artery pseudoaneurysm thirteen months following laparoscopic cholecystectomy. *EJVES Extra.* 2007;13:1–3.

25. Clements WD, Wilson RH, Crothers JG, et al. Pseudoaneurysm of the cystic artery following cholecystectomy. *J R Coll Surg Edinb.* 1993;38:348–349.

26. Mate AD, Surnare KR, Deolekar SS, Gvalani AK. Lower gastrointestinal bleeding due to hepatic artery pseudo- aneurysm following laparoscopic cholecystectomy. *J Minim Access Surg.* 2013;9(1):31–33.

27. Blocfl P, Modiano P, Foster D, Bouflot F, Gompel H. Recurrent haemobilia after laparoscopic cholecystectomy. *Surg Laparosc Endosc.* 1994;4:375–377.

28. Sebastián JJ, Peña E, Blas JM, Ceña G. Fatal upper gastrointestinal bleeding due to flepatic artery pseudoaneurysm diagnosed by endoscopy. *Dig Dis Sci.* 2008;53(4):1152–1153.

29. Machado NO, Al-Zadjali A, Kakaria AK, Younus S, Rahim MA, Al-Sukaiti R. Hepatic or cystic artery pseudoaneurysms following a laparoscopic cholecystectomy: literature review of aetiopathogenesis, presentation, diagnosis and management. *Sultan Qaboos Univ Med J.* 2017;17(2):e135-e146.

30. Soudack M, Epelman M, Gaitini D. Spontaneous thrombosis of hepatic posttraumatic pseudoaneurysms: sonographic and computed tomographic features. *J Ultrasound Med.* 2003;22-(1):99–103.

31. Feng W, Yue D, ZaiMing L, ZhaoYu L, Wei L, Qiyong G. Hemobilia following laparoscopic cholecystectomy: computed tomography findings and clinical outcome of transcatheter arterial embolization. *Acta Radiol.* 2017;58(1):46–52.

32. Wen F, Dong Y, Lu ZM, Liu ZY, Li W, Guo QY. Hemobilia after laparoscopic cholecystectomy: Imaging features and management of an unusual complication. *Surg Laparosc Endosc.* 2016;26(1):e18-e24.

33. Merrell SW, Scflneider PD. Hemobilia: evolution of current diagnosis and treatment. *West J Med.* 1991;155:621–625.

34. Kumar A, Sheikh A, Partyka L, Contractor S. Cystic artery pseudoaneurysm presenting as a complication of laparoscopic cholecystectomy treated with percutaneous thrombin injection. *Clin Imaging.* 2014;38:522–525.

35. Satoh H, Takeda T, Takashima M, Sumiyoshi K, Imaizumi N. Gas-forming liver abscess following transcatheter hepatic arterial embolization for an iatrogenic intrahepatic pseudoaneurysm: report of a case. *Surg. Today.* 1995;25(4):361–364.

36. Nicholson T, Travis S, Ettles D, et al. Hepatic artery angiography and embolization for flemobilia following laparoscopic cholecystectomy. *Cardiovasc Intervent Radiol.* 1999;22(1): 20–24.

37. Murugesan SD, Sathyanesan J, Lakshmanan A, et al. Massive flemobilia: a diagnostic and therapeutic challenge. *World J Surg.* 2014;38(7):1755–1762.

38. Hylton JR, Pevec WC. Successful treatment of an iatrogenic right hepatic artery pseudoaneurysm and stenosis with a stent graft. *J Vasc Surg.* 2010;51:1510–1513.

39. Davies O, Batt J, Bethune R, Courtney E. Hepatic artery pseudoaneurysm post laparoscopic cholecystectomy. *JSM Clin Case Rep.* 2014;2(5):1050.

40. Butet Y, Bouras AF, Truant S, Pruvot FR. Pseudoaneurysm of the cystic artery as a complication of laparoscopic cholecystectomy. *Dig Liver Dis.* 2012;44(5):449–450.